Amendments to the Specification

Please replace the paragraph at page 4, lines 4 through 32 with the following amended paragraph:

Tumor immunotherapy with humanized monoclonal antibodies (mAbs) such as HERCEPTIN® ~~Herceptin™~~ (trastuzumab) and RITUXAN® ~~Rituxan™~~ (rituximab) is now accepted clinical practice in patients with Her-2/neu$^+$ metastatic mammary carcinoma and B cell lymphoma, respectively (Wang, S. C., *et. al., Semin. Oncol.,* 28: 21-29, 2001; Leyland-Jones, B., *Lancet Oncol.,* 3: 137-144, 2002; Ranson, M. and M. X Sliwkowski, *Oncology,* 63 Suppl 1: 17-24 (2002), Johnson, P. and M. Glennie, *Semin. Oncol.,* 30: 3-8 (2003), Plosker, G. L. and D. P. Figgitt, Drugs, *63: 803-843 (2003)* and Ross, J. S., *et al., Am.* J. Clin. Pathol., 119: 472-485 (2003)). Based on their record of success, several other humanized mAbs are being developed and some, such as ERBITUX® ~~Erbitux™~~ (cetuximab) are apparently close to achieving final FDA approval. Nevertheless, antibody therapy is not uniformly effective, even in patients whose tumors express a high surface density of the target tumor antigen. Effector mechanisms thought to cause tumor regression are variable and particularly include inhibition of growth factor activity, as well as antibody-dependent cell-mediated cytotoxicity (ADCC). Complement-dependent cytotoxicity (CDC) has less frequently been identified as an effector mechanism and it remains somewhat controversial whether CDC contributes significantly to tumor regression. *In vitro* studies have shown that CDC is limited by membrane regulators of the complement system, such as CD55 and CD59, that are occasionally overexpressed on tumors. Moreover, the major complement-mediated effector mechanism used against microbial pathogens, C3-receptor-dependent phagocytosis and cytotoxic degranulation, is completely inactive against cancer. With the antitumor human IgG1-based mAbs that activate complement such as trastuzumab, rituximab, or cetuximab, a coating of iC3b is deposited on tumor cells that can be recognized by the leukocyte iC3b-receptor CR3 (Mac-1; CD11b/CD18; $\alpha_M\beta_2$-integrin). However, the triggering of CR3-dependent leukocyte (neutrophil, monocyte, macrophage, NK cell) mediated cytotoxicity requires that CR3 bind to both iC3b and binding to the lectin site. Since tumor cells do not express CR3-activating polysaccharides, they escape this protective mechanism effective against microbial pathogens.

Please replace the paragraph at page 32, lines 17 through 32 with the following amended paragraph:

Alternative to preparing monoclonal antibody-secreting hybridomas, a monoclonal antibody to a polypeptide of the invention can be identified and isolated by screening a recombinant combinatorial immunoglobulin library (*e.g.*, an antibody phage display library) with the polypeptide to thereby isolate immunoglobulin library members that bind the polypeptide. Kits for generating and screening phage display libraries are commercially available (*e.g.*, the Pharmacia *Recombinant Phage Antibody System*, Catalog No. 27-9400-01; and the Stratagene SURFZAP® ~~*SurfZAP*™~~ Phage Display Kit, Catalog No. 240612). Additionally, examples of methods and reagents particularly amenable for use in generating and screening antibody display library can be found in, for example, U.S. Patent No. 5,223,409; PCT Publication No. WO 92/18619; PCT Publication No. WO 91/17271; PCT Publication No. WO 92/20791; PCT Publication No. WO 92/15679; PCT Publication No. WO 93/01288; PCT Publication No. WO 92/01047; PCT Publication No. WO 92/09690; PCT Publication No. WO 90/02809; Fuchs *et al.* (1991) *Bio/Technology, 9*:1370-1372; Hay *et al.* (1992) *Hum. Antibod. Hybridomas, 3*:81-85; Huse *et al.* (1989) *Science, 246*:1275-1281; Griffiths *et al.* (1993) *EMBO J., 12*:725-734.

Please replace the paragraph at page 33, lines 6 through 15 with the following amended paragraph:

As illustrative of the inventive concept, β-glucans such as NSG could be administered to act synergistically with HERCEPTIN® ~~Herceptin™~~, a monoclonal antibody sold by Genentech for use in immunotherapy of breast cancer. HERCEPTIN® ~~Herceptin™~~ is a mAb that recognizes the her2 cell surface antigen which is present on 20% of breast cancer cell types. Clinical trials have demonstrated that HERCEPTIN® ~~Herceptin™~~ is saving lives, but its effectiveness could be significantly enhanced through concurrent administration of β-glucan. NSG therapy along with HERCEPTIN® ~~Herceptin™~~ therapy could result in a significant increase in the proportion of women responding to HERCEPTIN® ~~Herceptin™~~ therapy with long lasting remission of their breast cancer. Currently, only 15% of women receiving HERCEPTIN® ~~Herceptin™~~ therapy show long lasting remission.

Please replace the paragraph at page 58, lines 4 through 22 with the following amended paragraph:

In previous studies, β-glucan-mediated immunotherapy was thought to be enhanced by the coadministration of anti-tumor mAbs specific for a highly expressed and stable tumor antigen. The requirement that such mAbs activate complement was confirmed in experiments that demonstrated a failure of β-glucan to enhance mAb-mediated tumor regression or survival in C3-deficient mice. Others have also shown a lack of β-glucan enhancement of anti-tumor mAbs that did not activate complement (Cheung, N. K. and Modak, *Clin. Cancer Res.*, *8*: 1217-1223 (2002)). Thus, β-glucan cannot enhance the therapeutic activity of humanized mAbs that have been engineered in such a way that they do not activate complement. The majority of humanized mAbs containing the human IgG1 Fc-region have been shown to activate complement, such as HERCEPTIN® ~~Herceptin™~~, RITUXAN® ~~Rituxan™~~, and ERBITUX® ~~Erbitux™~~ (Spiridon, C. I., *et al., Clin. Cancer Res., 8*: 1720-1730 (2002), Idusogie, E. E., *et al., J. Immunol., 164*: 4178-4184 (2000), Cragg, M. S., *et al., Blood, 101*: 1045-1052 (2003), Herbst, R. S. and Hong, W. K., *Semin. Oncol., 29*: 18-30 (2002). With the exception of RITUXAN® ~~Rituxan~~, complement dependent cytotoxicity (CDC) does not represent a significant mechanism of tumoricidal activity with these mAbs and β-glucan does not alter the efficiency of CDC. Instead, β-glucan functions to prime granulocytes to kill tumor cells that have been targeted by mAb-mediated complement activation with surface-bound iC3b.